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The Lasting Damage to Mortality of Early-Life Adversity: Evidence from the English Famine of the late 1720s[♣]

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Abstract

This paper explores the long-term impact on mortality of exposure to early-life hardship. Using survival analysis, we document that birth during the great English famine of the late 1720s manifest itself in an increased death risk *throughout* life among those who survive the famine years. Using demographic data from the Cambridge Group's *Population History of England*, we find that the death risk of affected individuals who survived to age 10 is up to 66 percent higher than that of their control–group counterparts (those born in the five years following the famine). This corresponds to a loss of life-expectancy of more than 12 years. We find that effects differ geographically as well as with the socio-economic status of the household, with less well-off (manual-worker) families and families living in the English Midlands being hit the hardest. Evidence does not suggest, however, that children born in the five years *prior* to the famine suffered increased death risk.

JEL Codes: I12, J1, N33

Keywords: Death Risk, Malthus, Longevity, Positive Checks, Scarring Effect, Selection Effect.

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Introduction

The existence of *Malthusian positive checks* in pre-industrial England has been the subject of considerable interest in recent years.[1] While the magnitude of the *short-term* effects of hardship on deaths is heavily debated, no attention has been paid to the *long-term* effects: the influence of hardship on mortality later in life *among survivors*. The relevance of long-term effects has been brought into play by scholars of medicine and demography who hold that exposure to adverse condition in early life, such as famine and plague, has an impact on the subsequent mortality risk of the population by two opposing effects: a 'selection' effect whereby hardship kills off the weak, leaving only the strong, and thus potentially longer-lived, individuals to survive; and a 'scarring' effect where survivors suffer lasting damage to their vital organs and immune systems and, hence, incur augmented death risk throughout life.[2]

In this paper we use survival analysis to test the so-called 'fetal origins hypothesis' which holds that early-life under-nutrition leads to disproportionate growth *in utero* and infancy, which enhances susceptibility to illness and hence increases the death risk later in life (Barker 1995). We focus on the English famine of the late 1720s, the greatest of the eighteenth century. The data come from the Cambridge Group's *Population History of England from Family Reconstitution*, documented in Wrigley *et al*. (1997). An important advantage of this data is that individuals can be followed throughout life, allowing us to compute and compare the death risks and life-expectancies of cohorts born during the crisis years with cohorts born in adjacent years.

[1] See Nicolini (2007); Kelly and Ó Gráda (2010); Rathke and Sarferaz (2010); Møller and Sharp (2008).
[2] See Barker (1998); Bozzoli *et al*. (2009); Doblhammer (2004); Hatton (2011).

We use the Kaplan-Meier estimator of survival curves and the Cox Proportional Hazard Model to investigate effects. We look at cohorts born during each of the probable famine years 1727-30 as well as those born during the five years immediately preceding and the five years immediately following the famine. The estimates provide ample evidence that a 'scarring' effect dominated an eventual 'selection' effect in the two years identified as crisis years, 1727-1728, lending strong support to the 'fetal origins hypothesis'. More specifically, we find that children born during 1727-28 suffer an increased death risk and a lower life-expectancy *throughout* life compared to their control-group counterparts. Effects differ geographically and with the socio-economic status of the household, with less affluent (manual worker) families and families living in the English Midlands being hit the hardest. The death risk of affected individuals who survive to age 10 is up to 66 percent higher, and the life-expectancy up to 12 years lower, than in the control group.[3] The effect of early-life under-nutrition is also long-lasting: even at age 30 affected individuals face an amplified death risk of up to 71 percent, and a life-span up to 10 years shorter than their control-group counterparts. There is no evidence in the data, however, that individuals born in the five years *preceding* the famine suffer a significantly increased death risk later in life.

The rest of the paper explains how the results were derived. First, we describe in more detail the 'fetal origins hypothesis', and we portray the English famine of the late 1720s as it is reported in the existing literature. Then, we offer a more exact account of the data, the methodology used and the methodological

[3] As will become apparent below, the term 'death risk' here refers to the logarithm of the proportional hazard of a cohort relative to the control cohort.

issues that we encountered. Subsequently, we report, specify and discuss the results. Finally, we conclude.

Background

As is documented in details by Barker (1995, 1998) and Doblhammer (2004), individuals that are subject to undernourishment in very early stages of life are more likely to be diagnosed later in life with a wide range of illnesses – coronary heart disease, stroke, diabetes, chronic bronchitis etc.[4] The underlying view is that injuries caused during early childhood lie dormant until adulthood, or even old age, and are not clinically measureable before that point in time.

The mechanism by which disease experienced early in life affect the waiting time to the onset of illness are still unclear. But scholars seem to agree that exposure to undernourishment during periods when cell-growth is particularly rapid – especially *in utero* and infancy – can lead to long-lasting impairments of vital organs. Barker (1995, 1998) points out that the fetus is dependent on the nutrients from the mother and adapts to an inadequate nutrient supply by prioritization of brain growth at the expense of vital organs such as heart and lungs. He mentions that, although occurring in response to a transient phenomenon, these adaptations become permanent or 'programmed', resulting in irreparable abnormal constructions of vital organs and immune systems, which causes increased risk of autoimmune diseases and other illnesses at later stages in life.

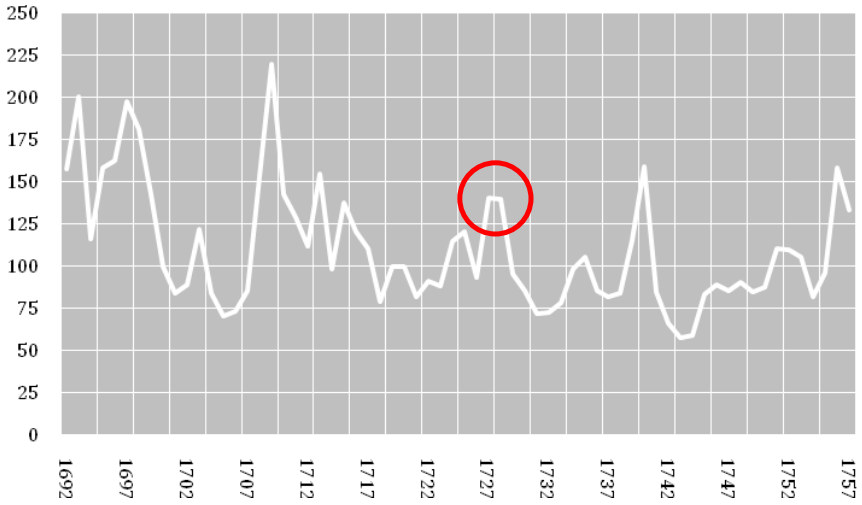
[4] Not all studies, however, are able to detect such effects. Kannisto and Christensen (1997), who look at cohorts born in Finland during the severe famine of 1866-1868, and Stanner *et al*. (1997), who look at cohorts born during the siege of Leningrad 1941-44, find little support for the 'fetal origins hypothesis'. Stanner *et al*. mention that one reason for their lacking effects is that malnutrition is necessary for prolonged periods. This conclusion would be consistent with our finding that effects are bigger among the poorer groups of society, as these are likely to be facing hardship also in non-famine years.

Periodic food shortage, and hence the risk of under-nutrition, was an unavoidable fact of life among ordinary people in pre-industrial times. Historical England was no exception: 'Until well into the nineteenth century no other aspect of economic life was consistently of such great concern to private individuals as to the public authorities alike as the scale of the last harvest and the prospects for the next year,' as Wrigley and Schofield (1989, 263) put it. Historical England witnessed several incidents of poor harvests, and thus scope for periodic starvation, in the run up to the industrial revolution. Summing up on these, Appleby (1980, 882) concludes that 'Of all the bad harvest years of the late seventeenth and early eighteenth centuries, 1727-28 is the only likely candidate for a subsistence crisis in England.'

Grain prices during those years were indeed in excess of their trend. Real wheat prices were 34 percent above the 25-year moving average in 1727; 61 percent above in 1728; and 29 percent above in 1729. It is clear from Figure 1 that the prices in 1727-28 were nowhere near their levels in the 1690s or in 1709-10.[5] But the years 1727-28 were also the only years between 1692 and 1757 when grain imports exceeded grain exports from England, as pointed out by Appleby (1980, 886).

[5] Real wages are nominal prices of wheat deflated by nominal agricultural day-wages from Allen (1998).

Figure 1:
Real Wheat Prices in England, 1692-1757 (Indexed: 100=1700)

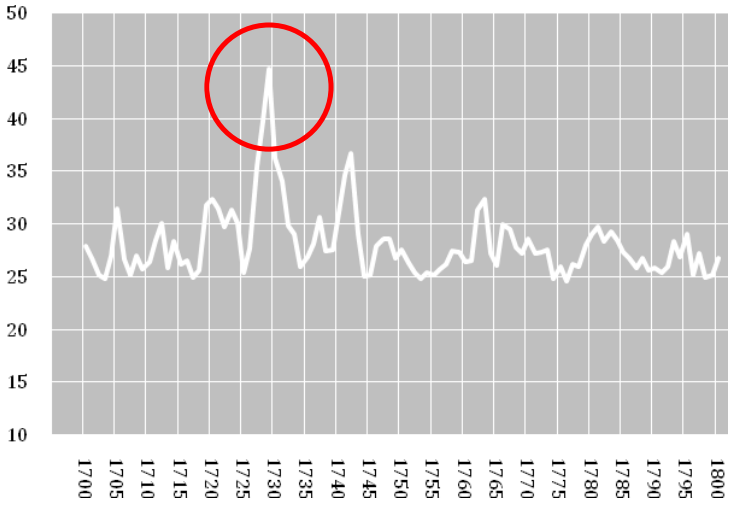


There is reason to believe, however, that increased food prices led to more than plain hunger. Indeed, before the twentieth century, most famine-related mortality was due to epidemic disease (Mokyr and Ó Gráda 2002; Ó Gráda 2007). Campbell (2009, 25) asserts that 'The heightened grain prices […] lend 1728 the appearance of a classic famine year, except that the death toll was heavier and net loss of population greater than is consistent with the scale of the price inflation and absolute level of real wages.' This led him to conclude that 'The demographic crisis of 1727–30 […] looks like a double disaster characterised by dearth and disease operating in tandem' (*ibid.,* 25).

Rather than looking at price-levels as an indicator of crisis, Wrigley and Schofield (1989, 332) have sought to classify crisis years by the degree of severity of death. Years where crude death rates were at least 10 percent above the 25-year moving average were categorized as years of crisis. Crisis years are further subdivided into three categories: a more than 10 percent deviation from the trend yields one star; more than 20 percent two stars: and more than 30

percent three stars. The median crude death rate in the period scrutinized by Wrigley and Schofield, i.e. 1541-1871, was 25.9 deaths per thousand. Death rates in 1727, 1728 and 1729, respectively, were 41.8, 43.2 and 42.2. In all three years there was a deviation of more than 35 percent from the 25-year moving average, placing the crisis of 1727-29 in the most brutal category: 3-star crisis. Figure 2 shows the spike in the crude deaths rates at the time. Although mortality was subject to local variation, as we shall see shortly, the crisis was deemed 'national' by Walter and Schofield (1991, 59) in the sense that over 28 percent of the 404 parishes analyzed showed excess rates of mortality.

Figure 2:
Crude death rates, England, 1700-1800



Data and Methodology

Below we proceed to analyse the effects on life-expectancy of being born during the famine of the late 1720s. Life-expectancy is the mean longevity of a given population, and longevity is the time interval between someone's birth and death dates. Birth and death dates of individuals, as well as their sex, location and

social background, come from the Cambridge Group's *Family Reconstitution* project, documented in Wrigley *et al*. (1997). This data is collected from the church books of a total of 26 parishes scattered across England in a way that make them representative of the entire country and includes the following locations: Aldenham, Alcester, Ash, Austrey, Banbury, Birstall, Bottesford, Bridford, Colyton, Dawlish, Earsdon, Gainsborough, Gedling, Great Oakley, Hartland, Ipplepen, Lowestoft, March, Methley, Morchard Bishop, Odiham, Shepshed, Southill, Reigate, Terling, and Willingham.[6]

In the Cambridge records it is very often the case that someone's birth and death dates are missing. As substitutes, demographers normally rely on baptism and burial dates instead. According to Wrigley and Schofield (1989, 96), the time-interval between birth and baptism dates was rarely more than one month (often less than two weeks), and the gap becomes smaller the further back in time one moves. Using baptism dates as a proxy for birth dates in the present case, therefore, does not seem to be a serious problem. As with baptisms versus births, burial dates are often reported in the church books instead of death dates. For obvious reasons, a burial normally took place within a few days after the time of someone's death, so the problem of inaccuracy in this regard is unlikely to have a significant impact on the results derived below. [7]

A more serious issue is that the Cambridge data is often censored in the sense that death/burial information is missing due to migration out of the parishes scrutinized (Souden 1984). Because the probability of migration increases with longevity, the mean longevity based on observations of birth and

[6] The data from the parishes Aldenham and Earsdon do not include any observations useful for the analysis.
[7] The proportion of burials in Hawkshed, Lancashire, in the late eighteenth century at different intervals after death were as follows: same day, 1%; 1st day, 21%; 2nd day, 50%; 3rd day, 25%; 4th day, 2%; 5th to 7th day, 1% (Schofield 1970).

death dates in the data is a downward-biased estimate of the mean longevity of the population. We tackle this issue with survival analysis, using the Kaplan–Meier Estimator of survival curves and the Cox Proportional Hazard Model. An important advantage of these methods are that they take into account the type of censoring which occurs if (as in medicine) a patient withdraws from a study, i.e. is lost from the sample before the final outcome is observed. This is exactly what happens in the current data.

More specifically, we use any information available that a censored individual is still alive at a given point in time, as revealed by the individual's marriage date or the births or deaths of siblings or parents. For instance, out of those whose death dates are censored, nearly 20 percent have their date of marriage available. The age at that date then acts as a lower bound for the length of their lives. For the remaining individuals whose death dates are censored, we adapt the following procedure. If their youngest sibling is born within 10 years after the individual, the time-interval between the individual's birth date and the birth date of their youngest sibling acts as a lower bound for the length of the individual's life. If the youngest sibling is born *after* 10 years, then 10 years is the lower bound for the length of life, on the assumption that the individuals did not move away from their family (and thus potentially out of the parish observed) before the age of 10. A similar approach is used regarding the death of the individual's mother, father or the youngest sibling that did not survive to the age of 10. These assumptions make it possible to estimate the survival curves of the various cohorts used in the analysis. Likewise, the hazard ratio between the crisis cohorts and the control-group can be calculated using the Cox Proportional Hazard Model.

There is some disagreement in the existing literature about when exactly the famine took place. Appleby (1980) believes that the crisis years included 1727-28; Wrigley and Schofield (1989) identifies 1727-29 as crisis years; and Campbell (2009) holds the crisis period to covered the years 1727-30. We therefore begin the analysis by looking at the cohorts born during each of the probable famine years 1727-30 and compare them to those born during the five years immediately preceding and the five years immediately following the famine.

The data used in the analysis contain a total of 12.640 individuals born in the period 1722-35. Among them, 53 percent were censored in terms of missing death dates. The individuals were divided into three main groups: those born in the five-year period *before* the crisis, 1722-1726; those born in one of the four potential crisis years, 1727-30; and those born in the five-year period to follow the crisis, 1731-35. The latter cohort performs as a control group. The reason for this is that the this cohort is exposed to similar macroeconomic conditions throughout life as the crisis cohorts, except for the fact that the post-crisis cohort is not exposed to the famine. Inspired by the 'fetal origins hypothesis', the *a priori* is that those among the crisis cohorts who survive the crisis will have an increased death risks, and thus a lower life-expectancy, compared to their control-group counterparts. The 'fetal origins hypothesis' would also imply, however, that those born *before* the crisis do not suffer lasting damage to mortality, because exposure to under-nourishment does not take place during periods when cell-growth is particularly rapid, i.e. *in utero* or infancy.

As concerns geography, Appleby (1980) and Wrigley and Schofield (1989) both mention that the famine struck mostly in the English Midlands, and

that the South-Western and the Northern parts of England were left more or less unharmed. Accordingly we subdivide the samples depending on whether the individual is born in a Midland or a non-Midland parish. Parishes situated in the Midlands include exactly half of the 26 locations, comprising Alcester, Austrey, Banbury, Bottesford, Gainsborough, Gedling, Great Oakley, Lowestoft, March, Shepshed, Southill, Terling, and Willingham.[8]

Finally, since it is also clear that the control-group individuals did not necessarily have the same socio-economic background as those born during the crisis, we use a two-step procedure to subdivide individuals into two groups, depending on the father's occupation. Firstly, documented in van Leeuwen *et al*. (2007), the so-called *History of Work Information System* (HISCO) gives standardized codes to hundreds of occupational titles existing in England between the sixteenth and the twentieth centuries. Using these codes in combination with the so-called HISCLASS system, documented in Van Leeuwen and Maas (2011), we are able to map all occupational titles in the data into one of two social classes: manual and non-manual labourers. Secondly, Clark and Hamilton (2006) have demonstrated, by analysing the wealth at death among male testators, that the wealth of manual labourers was significantly lower than that of their non-manual counterparts. Putting the HISCLASS and the wealth information together, we thus obtain a crude proxy for the wealth among those families in the data where the husband's occupation is available. Occupational data is available in 5,675 cases, or roughly half of the sample.

[8] We have experimented with a subdivision of parishes according to the elevation of the location. Subdividing parishes this way yields largely the same results as subdividing into Midlands and non-Midlands locations. The reason of this is that more than 80 percent of all individuals born in the Midlands are also born in a parish of low elevation.

Results

We begin by presenting the survival curves for the different cohorts. Survival curves capture the probability that an individual will survive beyond a specified age. According to the 'fetal origins hypothesis' cohorts subject to under-nutrition *in utero* or infancy will suffer increased death risk throughout life. In that case we would expect to see that the survival curves of the famine cohorts lie below the survival curve of the cohorts born in the five years immediately following the famine.

Plotted in Figure 3A-E are the estimated survival curves for the cohorts born in 1727, 1728, 1729 and 1730, respectively, as well as the pre-crisis cohort, displayed relative to the survival curve of the control group (i.e. those born during the period 1731-1735). The graphs give a clear impression that the cohorts born during 1728 and 1728 suffer an increased risk of dying throughout life compared to their control-group counterparts. The survival curves of those born during the years 1729 and 1730, on the other hand, do not seem to suffer from an increased risk of dying compared to the control group. Nor do the pre-crisis cohorts seem to differ from the post-crisis cohorts in terms of death risk.

Figure 3A:
Survival Curves: Pre-Crisis Cohort versus Control Group (Solid)

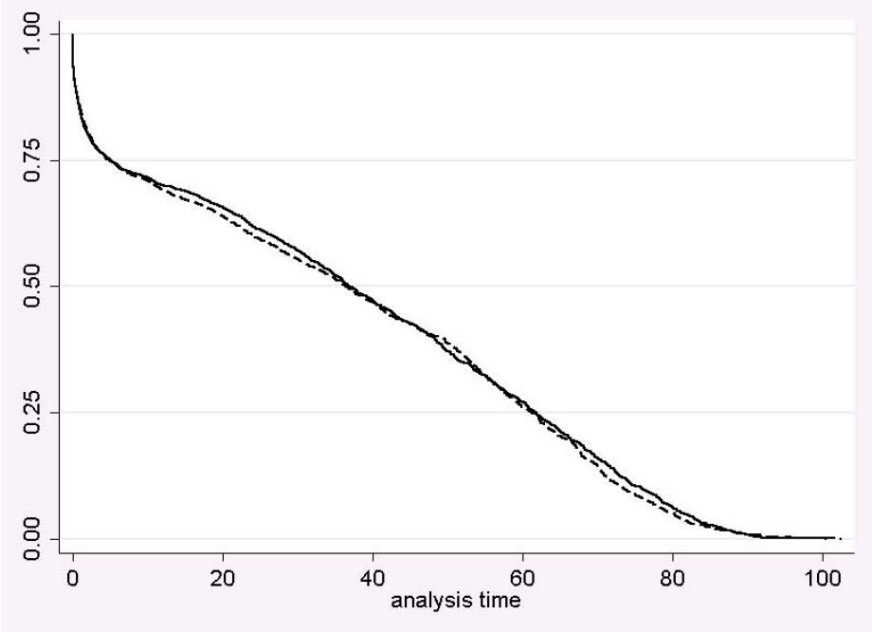


Figure 3B:
Survival Curves: 1727-Cohort versus Control Group (Solid)

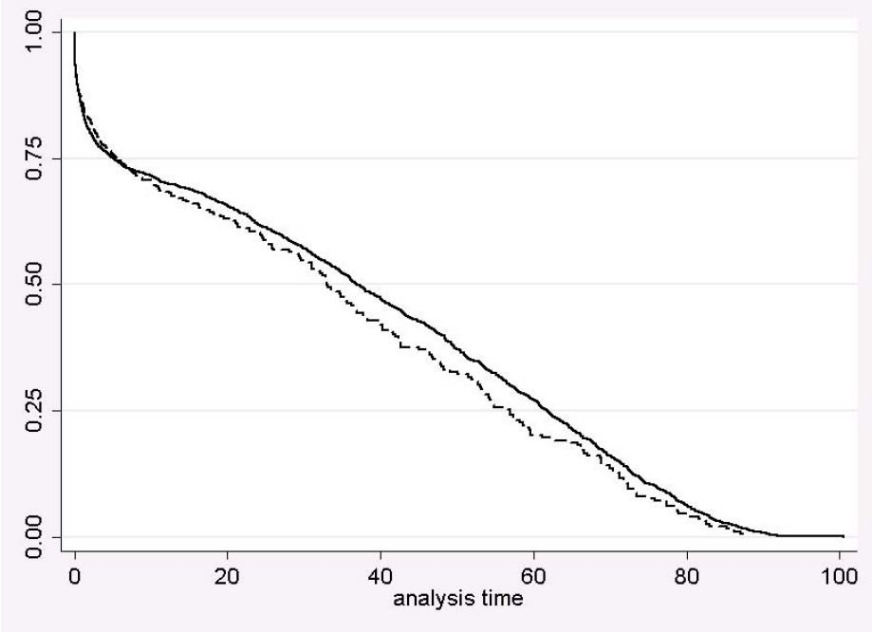


Figure 3C:
Survival Curves: 1728-Cohort versus Control Group (Solid)

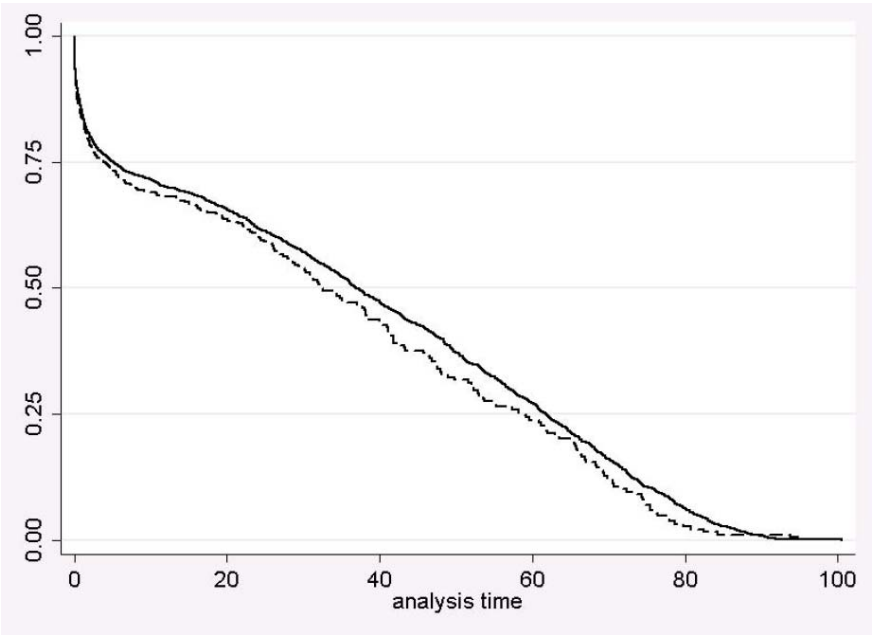


Figure 3D:
Survival Curves: 1729-Cohort versus Control Group (Solid)

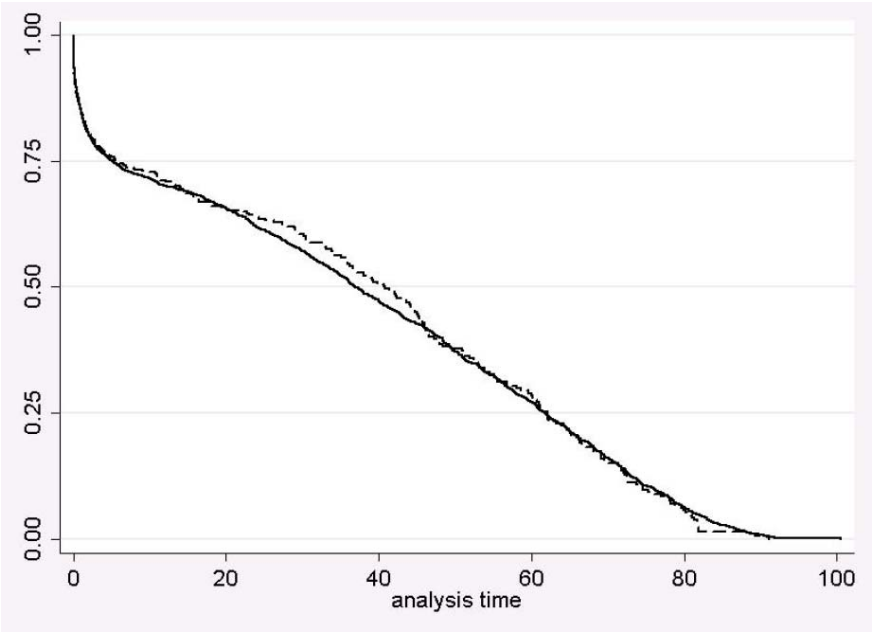
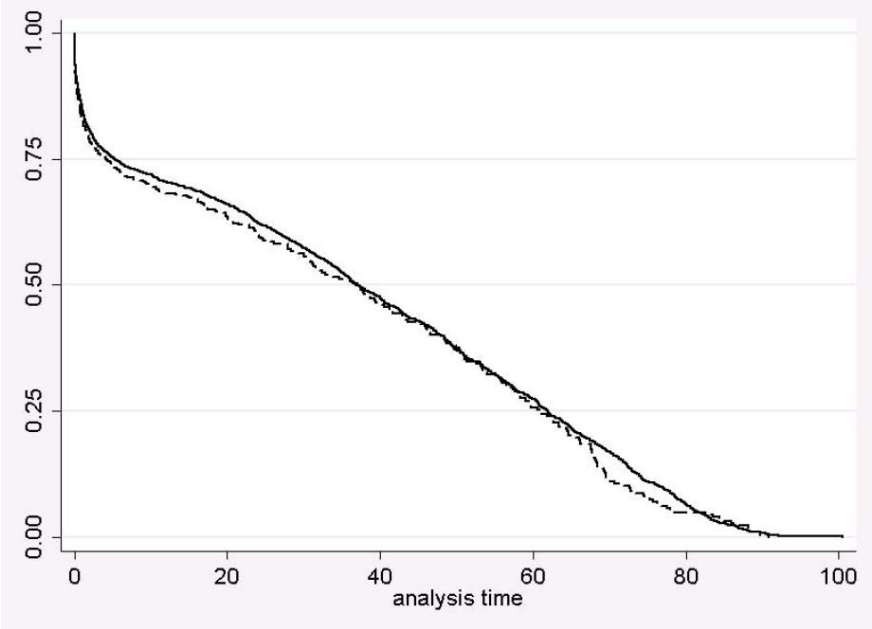


Figure 3E:
Survival Curves: 1730-Cohort versus Control Group (Solid)



These suppositions are backed up by the log-rank tests for equal survival distributions. The tests confirm that the survival curves of cohorts born in 1727 and 1728 are (in one case borderline) significantly different from that of the control group, when we look at each year separately (*p*-value equal to 0.101 for 1727 and to 0.0348 for 1728). Moreover, the combined crisis-cohort of 1727-1728 is significantly different from the control group (p=0.014). The survival curves of the 1729 and 1730 cohorts, however, are not statistically different from the control group's (*p*-values equal to 0.991 for 1729 and to 0.145 for 1730).

In order to treat the possibility of confounding variables, we use the Cox Proportional Hazard Model stratified by sex, birth order, Midlands location, and the father's occupation (a manual/non-manual dummy variable). Information

about the father's occupation was available in roughly 45 percent of the cases, so we also include a dummy for unknown occupation in the stratas.

Table 1:
Death Risks: All and by Region

Birth Cohorts	(1) All	(2) Non-Midlands	(3) Midlands
1722-1726	0.033 (0.037)	-0.018 (0.055)	0.075 (0.050)
1727	0.102 (0.063)	-0.069 (0.078)	0.290*** (0.098)
1728	0.151** (0.064)	-0.026 (0.089)	0.308*** (0.088)
1729	-0.025 (0.062)	-0.125 (0.089)	0.063 (0.089)
1730	0.0666 (-0.056)	-0.018 (0.094)	0.112 (0.083)
N	12,640	6,275	6,365

Robust standard errors clustered by family are used to calculate p-values. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level.

The results of the Cox Proportional Hazard Model are reported in Tables 1 and 2. All estimates report the logarithms of the hazard ratios. The numbers indicate to what extent the death risk of the pre-crisis and the crisis cohorts, i.e. those born during the period 1722-26 and during each of the years 1727 to 1730, compared to that of the control group. Positive numbers indicate an increased death risk vis-à-vis the control group; vice versa for negative numbers. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level. The number in the parenthesis is the standard deviation.

Table 2:
Death Risks: All and by Socio-Economic Group

Birth Cohorts	(1) All	(2) Non-Manual	(3) Manual
1722-1726	0.065 (0.052)	-0.006 (0.099)	0.089 (0.061)
1727	0.254** (0.113)	-0.335 (0.215)	0.458*** (0.110)
1728	0.360*** (0.094)	0.127 (0.180)	0.463*** (0.110)
1729	-0.074 (0.095)	-0.083 (0.165)	-0.084 (0.116)
1730	0.043 (0.089)	0.052 (0.148)	0.039 (0.107)
N	5,675	1,565	4,110

Robust standard errors clustered by family are used to calculate p-values. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level.

Column 1 of Table 1 shows that all cohorts, with the exception of those born during 1729, have an increased death risk compared to the control group. Only in the case of the 1728-cohort, however, is the death risk *significantly* higher. Furthermore, the subdivision of parishes into Midlands and non-Midlands (Columns 2 and 3) reveals – consistent with the *a priory* inspired by the existing literature – that only the Midland parishes were hit significantly. The subdivision also shows that the Midlands cohorts of 1727 and 1728 suffer a significantly higher death risk (roughly 30 percent) compared to their control-group counterparts. The remaining Midlands cohorts are also exposed to increased death risks, but the differences compared to the control group are not statistically significant.

The subsample of individuals for whom we have their father's occupation available (in 5,675 cases out of the 12,640 of the full sample) provides a picture largely identical to that of the full sample. Column 1 of Table 2 shows that the death risk of the cohorts born in 1727 and 1728 is significantly higher than their control-group counterparts, and that except for the 1729-cohort the remaining cohorts suffer slightly elevated, but not significantly higher, death risk relative to the control group. The most striking result appears when we subdivide individuals according to their father's occupation (manual versus non-manual labour). Column 2 shows that the death risk of the individuals of the non-manual households is not significantly higher than their control group counterparts. Column 3, on the other hand, demonstrates that the cohorts born in 1727 and 1728 are hit extremely hard by the famine, with a significantly increased death risk of roughly 45 percent compared to their control group.

Before proceeding any further, it is sensible to test the assumption of proportional hazards underlying the Cox Proportional Hazard Model. We have compared the plots of the scaled Schoenfeld residuals against age by each of the five cohort groups (1722-26, 1727, 1728, 1729 and 1730) for each of the six subsamples used in Tables 1 and 2 above. None of the plots raised doubts about the validity of the proportional hazards assumption. The null hypothesis of a zero slope cannot be rejected (even at the 10-precent level) in all cases but one. The rejected case is for the 1727-cohort in the non-Midlands parishes (p=0.038) for the full sample used in Table 1. The p-values of the global tests for non-zero slopes corresponding to the samples used in Columns 1, 2 and 3 of Table 1 were 0.95, 0.17 and 0.49, respectively, while the p-values corresponding to the

samples used in Columns 1, 2 and 3 of Table 2 were 0.80, 0.64 and 0.78, respectively.

The results of Tables 1 and 2 lead us to conclude that the most severe famine years are 1727-28, as these are the only years in which the death risks are significantly increased compared to the post-crisis control group. Hence, in the following we proceed to test the 'fetal origins hypothesis' for cohorts born during 1727 and 1728 relative to the control group. For parsimonious reasons, and in order to generate as many observations from the crisis cohorts as possible, we will consider the cohorts born during the years 1727-28 as one group, and then compare them to cohorts born during the post-crisis period. For consistency, we proceed to keep the cohorts born during the years 1731-35 as the control group.[9]

Table 3 reports the death-risk estimates of the 1727-28 cohorts at ages 0, 10, 20 and 30, respectively. The number below the parenthesis is the number of individuals in the 1727-28 cohort included in the regression. Column 1 reports the estimates using all observations, while Columns 2 and 3 subdivide observations into those born in the non-Midland and Midland parishes, respectively. It was clear from Tables 1 that the 1727-28 cohort was subject to increased death risk at age 0 (Table 3, first row). Yet, this could merely reflect the fact that death set in more or less immediately after the famine stroke, and that there were no long-term effects on mortality of those who survived the famine years. Column 1 of Table 3 shows, however, that individuals born during the famine years of 1727-28 suffer a statistically significantly increased death

[9] It does not make any qualitative difference to the conclusions obtained below if we use the cohorts born during the years 1730-1734 as a control group instead.

risk of more than 15 percent *also* at ages 10, 20 and 30. Meanwhile, consistent with the findings reported in Table 1 above, Columns 2 and 3 of Table 3 demonstrate that only individuals born in the Midlands parishes are subject to an increased death risk. Column 3 shows that the death risks are up to 28 percent higher when performing the analysis using only individuals born in the Midlands.

Table 3:
Death Risk at Various Ages of 1727-28 Cohort: All and by Region

Age	(1) All	(2) Non-Midlands	(3) Midlands
>0	0.125*** (0.049) 1,599	-0.050 (0.064) 877	0.299*** (0.070) 722
>10	0.158*** (0.072) 515	0.092 (0.093) 349	0.280** (0.111) 166
>20	0.150* (0.079) 455	0.099 (0.102) 315	0.255** (0.119) 140
>30	0.165* (0.085) 260	0.135 (0.190) 178	0.230* (0.133) 82

Robust standard errors clustered by family are used to calculate p-values. Bottom line numbers are number of observations in 1727-28 cohorts. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level.

When we look at the subsample of individuals about whom we have information about the father's occupation, the data clearly shows that *only* individuals born to less well-off families suffer a death risks that were significantly increased compared to their control-group counterparts. Judging by

the magnitude of the estimates, individuals born to more affluent families were also exposed to a higher death risk relative to their control group. But at the age of 10, the increased death risk of children of manual workers is nearly ten times bigger than that of their non-manual counterparts. This suggests that the rich are much less exposed to the famine conditions, and thus to its lasting impact, than the poor.

Table 4:
Death Risk at Various Ages of 1727-28 Cohort: All and by Socio-Economic Group

Age	(1) All	(2) Non-Manual	(3) Manual
>0	0.310***	-0.063	0.461***
	(0.077)	(0.155)	(0.085)
	584	168	416
>10	0.381***	0.067	0.529***
	(0.109)	(0.177)	(0.133)
	159	60	99
>20	0.381***	0.159	0.488***
	(0.125)	(0.208)	(0.192)
	132	51	40
>30	0.458***	0.370	0.518***
	(0.162)	(0.303)	(0.192)
	69	29	40

Robust standard errors clustered by family are used to calculate p-values. Bottom line numbers are number of observations in 1727-28 cohorts. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level.

That conclusion is interesting to parallel with work by Kelly and Ó Gráda (2010). Their data concerns a very different time period, namely the eighty years leading up to the Black Death in 1348, and they study only the short-term effects

of hardship. Nevertheless, they find that poor harvests are deadly at *both* ends of society, with tenants dying immediately and nobles with a lag of a year.

Table 5:
Death Risk 1727-28 Cohort in Non-Midlands: All and by Socio-Economic Group

Age	(1) All	(2) Non-Manual	(3) Manual
>0	0.002	-0.224	0.173
	(0.137)	(0.240)	(0.145)
	186	91	95
>10	0.083	-0.211	0.279
	(0.172)	(0.251)	(0.234)
	74	40	34
>20	0.082	-0.198	0.250
	(0.208)	(0.324)	(0.271)
	64	34	30
>30	0.076	-0.031	0.154
	(0.272)	(0.483)	(0.317)
	34	18	16

Robust standard errors clustered by family are used to calculate p-values. Bottom line numbers are number of observations in 1727-28 cohorts. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level.

Given what we now know about the individuals whose fathers were manual workers, an interesting question is whether the Midlands were hit harder by the famine because its parishes contained more manual-worker families, or whether it had to do with geography (or both). Together Tables 5 and 6 can shed light on the issue, keeping in mind that the number of observations become rather low, especially as we move into later stages of life. Table 5 shows the death risk of individuals born in a non-Midlands parish. Here, it is clear that, while individuals from manual-worker families suffer an increased death risk,

none of the individuals of the two socio-economic groups were hit significantly by the famine (although this could be a matter of a small number of observations).

Table 6:
Death Risk 1727-28 Cohort in Midlands: All and by Socio-Economic Group

Age	(1) All	(2) Non-Manual	(3) Manual
>0	0.417***	0.025	0.527***
	(0.090)	(0.204)	(0.096)
	398	77	321
>10	0.590***	0.378	0.660***
	(0.135)	(0.246)	(0.154)
	85	20	65
>20	0.606***	0.561***	0.630***
	(0.153)	(0.246)	(0.184)
	68	17	51
>30	0.730***	0.844**	0.711***
	(0.208)	(0.356)	(0.247)
	35	11	24

Robust standard errors clustered by family are used to calculate p-values. Bottom line numbers are number of observations in 1727-28 cohorts. One, two and three stars indicate statistical significance at the 10%, 5% and 1% level.

By contrast, Table 6 shows that individuals born in a Midlands parish are significantly worse off than their control-group counterparts, particularly later in life. Judging merely by the *magnitude* of the estimates, the conclusion is similar in the sense that the death risks of manual workers in the Midlands parishes is twice as big as that of their non-Midlands counterparts. Likewise, it is clear that the Midlands individuals from non-manual families are subject to a substantially increased death risk compared to their non-Midlands equivalents (who appear

to have reduced death risks compared to their control-group counterparts). In summary, the key message to take away from Tables 3-6 is that those who where hit the hardest – individuals of poor families in the Midlands area – suffer an increased death risk throughout life of over 60 percent.

Another interesting question is how big the loss in terms of life-expectancy was among the various groups at different stages of life. These results are reported in Table 7. The estimates – i.e. the number of years lost among the individuals of the crisis cohort – are based on differences in the restricted means between the 1727-28 cohort and their control-group counterparts. While the overall loss of life-expectancy among all individuals in the sample is 2.7 years (Column 1), it is clear that number hides a lot of information about geographic and socio-economic differences in the population. The biggest effect is found among individuals born to poor (manual worker) families in the Midlands, showing that the average loss of life at age 10 is more than 12 years compared to the control group. Given that the life-expectancy at age 10 among control-group individuals is 40 years (meaning that they live to reach age 50 on average), the life-expectancy of an affected individual is 25 percent shorter – a substantial loss of life.

Table 7:
Differences in Life-Expectancy at Age 10: 1727-28 Cohort versus Control Group

N=12,640	(1) All	(2) Non-Midlands	(3) Midlands
All	-2.7	-2.3	-4.3
N=5,675	All	Non-Manual	Manual
All	-6.4	-2.4	-8.5
Non-Midlands	-3.8	-4.3	-2.8
Midlands	-9.2	1.3	-12.5

Differences are based on the estimates of the mean longevity obtained as the integral of the survival functions estimated with the Kaplan-Meier estimator for the 1727-28 cohorts and the control-group cohorts.

Conclusion

Using demographic data from the Cambridge Group's *Population History of England*, this study documents that individuals born during the famine of the late 1720s suffer markedly higher death risk and considerably lower life-expectancy compared to their control-group counterparts, not only at birth but also later in life. This suggests that a 'scarring' effect was dominating an eventual 'selection' effect, lending strong support to the 'fetal origins hypothesis' proposed by Barker (1995).

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